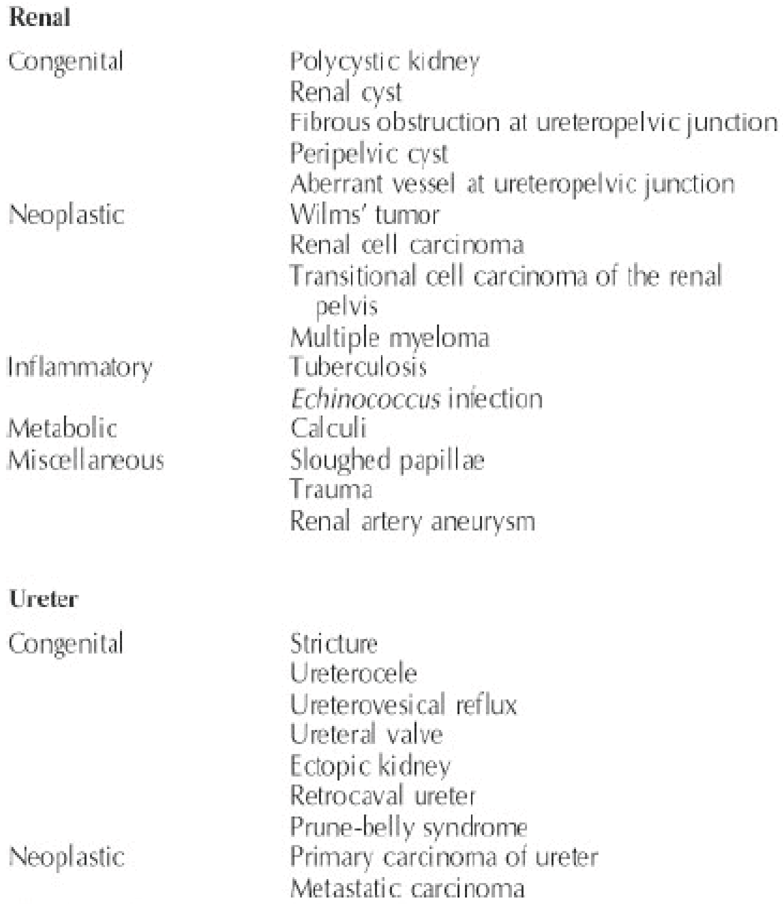
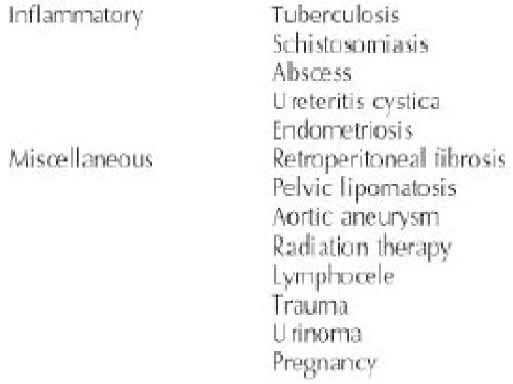
Pathophysiology of Urinary Tract Obstruction

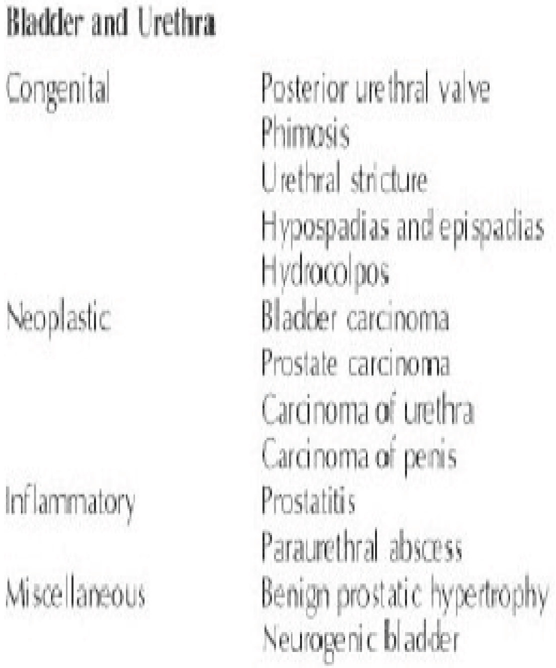
Causes of obstructive nephropathy



Renal	
Congenital	Polycystic kidney Renal cyst Fibrous obstruction at ureteropelvic junction Peripelvic cyst Aberrant vessel at ureteropelvic junction
Neoplastic	Wilms' tumor Renal cell carcinoma Transitional cell carcinoma of the renal pelvis Multiple myeloma
Inflammatory	Tuberculosis *Echinococcus* infection
Metabolic	Calculi
Miscellaneous	Sloughed papillae Trauma Renal artery aneurysm
Ureter	
Congenital	Stricture Ureterocele Ureterovesical reflux Ureteral valve Ectopic kidney Retrocaval ureter Prune-belly syndrome
Neoplastic	Primary carcinoma of ureter Metastatic carcinoma



Inflammatory	Tuberculosis Schistosomiasis Abscess Ureteritis cystica Endometriosis
Miscellaneous	Retroperitoneal fibrosis Pelvic lipomatosis Aortic aneurysm Radiation therapy Lymphocele Trauma Urinoma Pregnancy



Bladder and Urethra	
Congenital	Posterior urethral valve Phimosis Urethral stricture Hypospadias and epispadias Hydrocolpos
Neoplastic	Bladder carcinoma Prostate carcinoma Carcinoma of urethra Carcinoma of penis
Inflammatory	Prostatitis Paraurethral abscess
Miscellaneous	Benign prostatic hypertrophy Neurogenic bladder

renal function change in response to obstruction

1- in Unilateral Ureteric Occlusion (UUO) there is TRIPHASIC response

	Phase 1	Phase 2	Phase 3
Time	(1-2hours)	(2-5hours)	(>5hours)
Etiology	early renal VD from **PGE2 and NO**	efferent V.C (mediated by **AT2, TXA2, and endothelin**)	afferent VC & decreasing filtration
RBF	Increase ↑	decreasing↓	Decreasing↓
uretral pressure	increase↑	increase↑	Decreasing↓
GFR	Stable d2 (tubule glomerular feedback)	decreasing	decreasing

2- in Bilateral Ureteric Occlusion (BUO) there is BIPHASIC response

	Phase 1	Phase 2 a	Phase 2 b
Time	(<1hour)	(1-5hours)	(>5hours)
Etiology	As above		
RBF	*mildly* ↑'d	↓'ing	↓↓↓'ing
uretral pressure	↑↑↑'d	↑'d	↑'d (x24hrs)*
GFR	decreasing	decreasing	decreasing

***ureteral pressure stays elevated due to accumulation of vasoactive substances (eg ANP) accounts for difference from UUO**

After release of BUO

- GFR and RBF remain depressed due to persistent vasoconstriction of afferent arteriole
- ↑'d urine flow + Na excretion mediated by expanded volume, osmotic agents (eg urea) & ANP

The effects of obstruction

1- on ureteral function

the effect of obstruction depends on degree & duration of obstruction, rate of urine flow, and presence of infection

- increased resting ureteral intraluminal pressure initially reaches maximum ~3hrs then declines afterwards to a little above baseline, then plateaus
  - due to reduced RBF, GFR, pyelovenous reabsorption, etc
- transient ↑ in amplitude & frequency of peristaltic ctx's
- gradual increase in ureteral length & diameter potential for ↑'d contractility offset by dilated, thin muscle
- ureteral smooth muscle hypertrophy
- presence of infection decreasing ureteral contractions so impairs urine transport
- obstructed ureter can't coapt ureteral wall & can't generate N active intraluminal pressures

N ureteral peristalsis can occur after relief of obstruction <2wks

2- on renal tubule function

1) lower GFR
2) ↓'d urine concentrating ability (decreased urine osmolality) due to
   - loss of active salt reabsorption from ThickAL and a reduction in Na transporters
   - loss of urea backflux from inner medullary CD
   - decreased water reabsorption in PCT, ThinAL, and CD
3) increased Fractional Excretion of Sodium FENa ・ profound in BUO, mild with UUO

*** in acute obstruction, see opposite, increased urine osmolality and decreased FENa

3- on Cellular & molecular changes

1) **tubulointerstitial fibrosis**
   - accumulation of extracellular matrix from increased synthesis of tissue inhibitors of metalloproteinases (**TIMPs**)
   - stimulation of inflammatory cytokines (**TGF-β, ATII, NFκB, & TNF-α)**
2) **tubular atrophy & apoptosis**
   - starts after day 4 (mediated by **caspases , TNF-α**)
   - glomerular cells are resistant to this type of apoptosis
3) **interstitial inflammation**

pathologic changes seen with obstruction after 6 weeks of obstruction

1) gross pathology

- pelvicaliectasis
- initially heavier more edematous parenchyma but becomes LIGHTER than contralateral kidney 6 weeks after obstruction
- larger in size
- cystic appearance

2) microscopic pathology

- glomerular collapse
- tubular atrophy
- more prominent collagen and elastin in outer cortex and outer medulla (role of TGF-β)
- hyalinization & proliferation of connective tissue in collecting system
- lymphatic dilation
- interstitial edema and fibrosis
- macrophage infiltration
- hemorrhage and necrosis
- renal pelvic smooth muscle atrophy

Drugs can be used to try to decrease renal fibrosis and functional impairment

- ACE inhibitors ・ decreased ATII-mediated fibrosis and apoptosis of renal tubules
- aldosterone antagonist (eg spironolactone) ・ decreased aldosterone-mediated fibrosis
- downregulation of NO (L-arginine) ・ decreased NOS-mediated inflammation

compensatory renal growth

- compensatory growth (hyperplastic + hypertrophic) seen in contralateral kidney
- NO increase in # of nephrons or glomeruli
- **mediated by IGF-1**
- factor affect compensatory renal growth
    - age } less seen in older patients
    - degree of obstruction } less prominent with partial UUO
    - duration of obstruction } more with longer durations of UUO

renal recovery after obstruction relief

- usually get **full recovery if <2 weeks of obstruction**
- little to **no recovery after >6weeks of obstruction**
    - the longer the obstruction, the less likely the return to normal GFR
- 2 phases of functional improvement
    - **first 2 weeks ・ improved tubular function**
    - **the next 10 weeks ・ gradual improvement of GFR**

- **predictors of renal function recovery after obstruction**
  - short duration
  - smaller degree of obstruction
  - greater compliance of collecting system
  - presence of pyelolymphatic backflow
  - younger age
  - no renal cortical thinning
  - normal baseline renal function
    - renal scan **DMSA best at predicting renal recovery**

5 different paths of urine egression in an obstructed kidney

1) forniceal rupture
2) pyelosinus
3) pyelolymphatic
4) pyelovenous
5) pyelotubular

MANAGEMENT OF PATIENTS WITH OBSTRUCTION

A. Imaging

1- U/S “**1st line”**

- no radiation, no contrast, cheap, safe in pregnancy and kids
- mainly anatomic info only can assess cortical thickness (chronicity of obstruction)
- can add Doppler to assess functional component } elevated RI + hydro may represent high-grade obstruction
- FALSE +VE hydro may not represent obstruction
  - capacious extrarenal pelvis
  - parapelvic cysts
  - VUR
  - high urine flow rate
  - full bladder/retention
- FALSE –VE obstruction may not have hydro
  - acute (prior to developmenthydronephrosis
  - intrarenal collecting system
  - dehydration
  - misinterpretation of caliectasis as renal cortical cysts
  - forniceal rupture

2- **IVP “old gold standard”**

- **Give anatomic AND functional info**
- requires contrast and radiation
- delayed nephrogram and pyelogram indicates functional abnormality
- delayed images give anatomic info (ie hydro, level of obstruction, ureteral tortuosity)
- signs of obstruction on IVP?
    - ▪ acute
        - delay in calyceal filling
        - dilation of collecting system
        - nephromegaly
        - obstructive nephrogram
        - fornix rupture with extravasation of contrast
    - ▪ chronic
        - ureteral dilation & tortuosity
        - parenchymal thinning
        - calyceal crescents
        - soap-bubble nephrogram
        - standing column of contrast

3- retrograde pyelogram

- ○ anatomic info only may give more detail than other
- ○ safe with renal insufficiency and contrast allergy

4- antegrade pyelography

- **indication**
    - when retrograde pyelography not feasible
    - when other imaging studies don't adequately define collecting system or ureteral anatomy

5- Whitaker test

- limited use "invasive and often discordant results"
- measurement of renal pelvic pressure during infusion of saline or contrast into collecting system via NT or perc needle at a fixed rate of 10cc/min
    - **intrapelvic pressure <15cm H2O = normal**
    - **intrapelvic pressure 15-22cm H2O = indeterminate**
    - **Intrapelvic pressure >22cm H2O = obstruction**
- true renal pelvic pressure = collecting system pressure – intravesical pressure
- extrinsic factors affect pressure readings during a Whitaker test
    - needle size and flow rate - tube length and compliance
    - viscosity of perfusing fluid - temperature
    - presence of catheter/fullness of bladder

6- renal scans

- noninvasive, no contrast, no radiation
- radioactive tracers are given iv and used to evaluate functional and obstruction
    - **T1/2 <10 minutes = normal**
    - **T1/2 >20 minutes = obstruction**
    - **T1/2 10-20 minutes = indeterminate**
-

- **Precaution**
    - hydrate all patients
    - give lasix to produce high-flow and r/o dilated but unobstructed system
        - standard is F+20 study
        - if equivocal, do an F-0 or F-15 study
            - normalizes partially obstructed-looking curve
    - empty bladder prior to renal scan and Foley if can't void it is indicated in
            - suspected LUT obstruction
            - VUR
            - neurogenic or non-compliant bladder
            - low-lying, pelvic kidneys
- **causes of a falsely delayed renal scan**
        - capacious collecting system
        - renal insufficiency
        - renal immaturity in neonates
        - dehydration
        - presence of VUR
        - elevated intravesical pressure (full bladder, neurogenic bladder, inability to void, etc)
        - patient motion

- **the main tracers used**

	DTPA diethylenetriaminepentacetic acid	**DMSA** dimercaptosuccinic acid	**MAG3** mercaptoacetyltriglycin
	glomerular tracer	cortical-tubular tracer	tubular tracer
Character	100% filtration	65% secretion + 35% filtration cortical retention	100%tubular secretion (in PCT) little cortical retention
value in GFR	best measure of GFR	gives information on GFR best assessing parenchyma (scars) and split function	more accurate numerical **GFR**
Value In obstruction	good to measure obstruction	Not good	more accurate numerical **obstruction**
OTHER	not good when kidney function is low or immature as 20% extraction coefficient in mature kidney		accurate when kidney function is poor or immature(50% extraction co efficient in mature kidney)

NB: ways to determine split/differential renal function

- nuclear renography
- split 24-hr urine creatinine clearance
- split 24-hr urine inulin clearance

7- CT

- **1st line for suspected ureteral obstruction**
    - shows more anatomic detail than U/S and IVP
    - shows some functional info when used with contrast enhancement
    - non-contrast spiral CT 97% accurate for ureteral stones can see radiolucent stones
- signs of obstruction
    - ureteral dilation
    - perinephric stranding
    - perinephric fluid
    - nephromegaly

8- MRI

- Advantages
    - no radiation or contrast so safe in pregnancy
    - accurate to dx obstruction
- disadvantages
    - high cost, longer time to do study
    - CAN'T identify ureteral stones
    - Can't identify ureteral anatomy in unobstructed system
        - add lasix to help identify ureteral anatomy in unobstructed system
- indication
    - pts w/ renal failure
    - pts w/ contrast allergy

B. Pain Management

using NSAIDs in controlling pain

- benefit
  - better pain reduction, less need for rescue analgesia & less N/V than narcotics
  - decreased PG-induced pain potentiation
  - decreased renal pelvic pressures by decreasing RBF (vasoconstriction of afferent arteriole)
  - decreased renal pelvic pressures by up-regulating Aquaporin 2 (AQP2) channels which reabsorb water
- NOT for patients with renal insufficiency

C. Surgical Intervention

- **indications to urgently relieve obstruction**
  - unilateral obstruction
      - intractable pain or N/V
      - signs of sepsis
      - high-grade obstruction
  - bilateral obstruction
    - same as UUO + the following
      - elevated BUN/creatinine or K+
      - signs & symptoms of uremia
      - signs & symptoms of fluid overload

- **ureteral stents or NTs**
  - no significant difference in health-related QOL
  - stent better in coagulopathic patients
  - NTs better in cases of extrinsic obstruction as
    - more reliable decompression w/ larger caliber instrument (eg 8Fr pigtail vs 6Fr stent)

- **consider Nx over reconstruction for obstructive uropathy**
  - first drain kidney for at least 6-8 weeks before renal scan
  - if differential renal function <10-15% consider nephrectomy
  - in patients with renal dysfunction, may want to leave kidney even if <10-15% function as it may prolong the time until need for dialysis

Post-Obstructive Diuresis

Define

urine output >200cc/hr following relief of urinary tract obstruction only with BUO or UUO of solitary kidney

Causes

1) **Physiologic POD** most common cause
    - volume overload (↑’d fluid and ANP)
    - solute accumulation (Na, urea)

2) **Iatrogenic POD**
    - excessive re-hydration
    - use of glucose-containing fluid (osmotic diuresis)

3) **Pathologic POD** due to several factors:

    a) loss of active Na reabsorption from ThinAL “salt-wasting nephropathy” mediated by ANP & ↓’d Na transporters (ENaC, Na/K/ATPase, Na/K/2Cl)

    b) loss of urea backflux from inner medullary CD “medullary washout”

    c) decreased H2O reabsorption in PCT, ThickAL, and CD poor response to ADH mediated by down-regulation of AQP-2 channels “nephrogenic DI”

Pathologic POD usually only occurs in patients with:

- signs of fluid overload (edema, CHF, HTN)
- abnormal renal function
- abnormal electrolytes

RFs for POD = occurs of pathological POD

- clinical
    - chronic obstruction
    - HTN
    - CHF
    - edema
    - wt gain
    - DM
    - uremic encephalopathy
- laboratory
    - azotemia
    - hyperK
    - hyperPO4
    - hypoCa
    - low bicarb (acidosis)

Electrolyte abnormalities are commonly seen POD

- acidosis
- hypoNa
- hypoK (can be elevated if impaired renal function)
- hypoPO4
- hypoMg

Management of POD

1) N renal function + lytes, no fluid overload, mentally alert and able to drink

- monitor vitals & U/O
- continue to drink & monitor VS's + daily lytes, Mg, PO4, creatinine until POD resolves

2) abN renal fxn or lytes, fluid overload, poor cognitive function or hypovolemic

- admit and monitor vitals and U/O q2-4h
- monitor serum lytes and serum creatinine q12h or less
- urine electrolytes (guides replacement fluids) and urine osmolality
- iv fluids replaced at 1:2 } type of fluid based on urine lytes (start with 1/2 NS)

causes of polyuria

1) Water diuresis

- decreased ADH
    - ADH inhibitors } α-agonists, EtOH, opioids, Dilantin
    - excessive intake of hypotonic fluid
    - psychogenic polydipsia
- impaired response to ADH
    - nephrogenic DI ("OLD CARP FISH")
        - obstruction, Lithium, Demeclocycline
        - Ca excess, Ampho B, Renal failure, PCKD
        - Familial X-linked disorder, idiopathic, sickle cell disease, hypoK
    - cold diuresis
- thirst
    - hyperCa
    - hyper-reninemia
    - K depletion
    - organic primary polydipsia
- central DI
    - idiopathic
    - neoplastic, infiltrative, vascular, inflammatory, trauma

2) Solute diuresis

- Nonelectrolyte
  - Urea
    - metabolic :hypercatabolism, high protein diet
    - renal disease :POD, diuretic phase of ATN, post-Tx diuresis
  - Glucose DM, renal glucosuria, D5W infusion
  - Mannitol
  - Glycerol
  - amino acids
- Electrolyte
  - NS infusion or ++ salt intake
  - diuretics
  - increased ANP secretion
  - hypoaldosteronism
  - renal disease } impaired tubular reabsorption
    - salt-losing nephritis
    - POD
    - diuretic phase of ATN
    - post-TX diuresis
    - chronic tubulointerstitial disease

3) Mixed water-solute diuresis

- combined uncontrolled DM and CRF
- POD

Retroperitoneal Fibrosis aka Ormond's disease, periureteritis fibrosa, fibrous retroperitonitis

It is condition in which a predominantly inflammatory mass in the retroperitoneum envelops & potentially obstructs retroperitoneal structures

incidence

- 1 in 200,000
- 3:1 M predominance
- mean age of onset is 50yrs

pathology

- usually at ~L4/L5 level
- gross appearance
    - fibrous, whitish plaque encasing
        - aorta, IVC, and their major branches,
        - ureters, & other retroperitoneal structures
        - may also involve intraperitoneal structures including the GIT
- **occurs in 2 phases:**
    - **inflammatory phase**
        - plasma cells, lymphocytes, macrophages, and eosinophils
        - likely an autoimmune response (?leakage of ceroid lipoprotein from atheromatous plaque in aorta)
        - inflammation more intense at margins of mass
    - **fibrotic maturation phase**
        - development of homogeneous fibrous tissue w/ limited cellularity

Etiology

- idiopathic 70 %
- retroperitoneal malignancy “10%”
- others
  - Radiation
  - Periarteritis (eg AAA, collagen vascular disease , inflammatory response to advance athrosclerosis)
  - Trauma
  - Infection (eg syphilis, TB, gonorrhea, UTI etc)
  - Inflammatory (eg IBD, ascending lymphangitis)
  - Chemical
    - Talc powder
  - Hemorrhage
    - Abdominal and pelvic surgery
    - Rupture viscus
  - Other
    - Biliary disease
    - Sarcoidosis
    - Endometriosis
  - Meds
    - β-blockers - Hydralazine - Amphetamines
    - Ergots - Haldol - LSD
    - Methyldopa and Methylsergide
    - Reserpine

Presentation

- usually presents with nonspecific signs and symptoms
    - fever , anorexia, malaise , N/V , wt loss -
    - HTN (50%)
    - back, abdo, flank pain
    - oliguria/anuria
    - lower limb edema – DVT from vascular compression

investigation

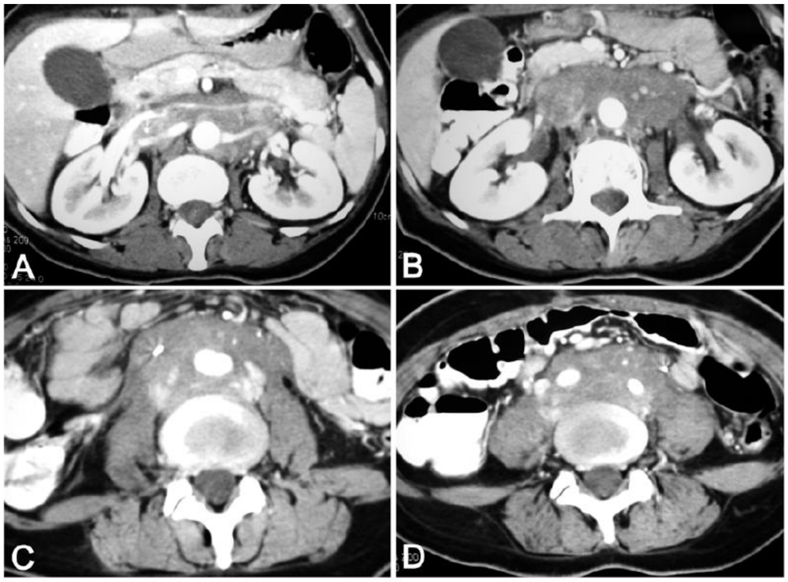
1- lab
    - **elevated ESR (90% - most common)** mainly in inflammatory phase
    - anemia
    - high creatinine
    - hyergammaglobulinemia

2- U/S
    - **hydronephrosis (98%)** from extrinsic compression

3- classic IVP triad
    - medially deviated ureters (nonspecific)
    - extrinsic ureteric compression smooth, hypoechoic mass anterior to lumbar or sacral spine
    - proximal hydroureteronephrosis

4- **CT “**modality of choice”

- In non contract CT
    - i. fibrotic plaque has similar HU as muscle
    - ii. can’t distinguish from great vessels
- In CT with contrast
    - i. contrast enhancement of mass common (esp. in areas of active inflammatory fibrosis)
    - ii. symmetric, continuous encasement of aorta and IVC
    - iii. no evidence of primary malignancies + no significant lymphadenopathy



5- **MRI**

- hypointense mass compared to muscle on T1 MRI with variable GAD enhancement more enhancement in acute phase
    - i. better discrimination of soft tissue
    - ii. can distinguish from great vessels

management

1) ACUTE MANAGEMENT

- r/o malignancies
- stop any possible offending meds spontaneous resolution is rare
- preservation of renal fxn
    - prompt decompression with NT or JJ stent usually JJ stent goes easily
    - monitor for post-obstructive diuresis

2) MEDICAL MANAGEMENT (**"STAMP-C"**)

- **Steroids**
    - r/o malignancy prior to initiating Rx
    - prednisolone 20-60mg PO q2days x 6-8wks, then tapered down to 5mg PO OD
    - those w/ systemic manifestions or lab evidence of active inflammation tend to respond better
- **Tamoxifen**
    - anti-estrogen effects on fibrous tissue (?exact mechanism of action)
    - 20mg PO OD x 12 months (monotherapy)
        - mainly used after failure of steroids
        - can also be taken after steroid Rx
- **immunosuppressants**
    - used in combo with steroids or as monotherapy
    - **A**ZT (2.5mg/kg PO OD x 3-6mos, then 1.5mg/kg PO OD x 6mos)
    - **C**yclophosphamide (2mg/kd po OD x 3mos, then taper off by 6 mos)
    - **P**enicillamine & **M**MF have also been used

3) SURGICAL MANAGEMENT

- 2nd line therapy, after trial of medical management
- always consider treatment of both ureters, even if disease involves 1 ureter
    - open Bx +/- ureterolysis
        - deep Bx sent for frozen and permanent sections
    - start dissection at distal non-dilated ureter
    - mobilize ureter off mass
    - displace anteriorly into peritoneal cavity wrap in omentum or PTFE & close peritoneum behind
    - consider post-op steroids to decrease recurrence
- laparoscopic Bx +/- ureterolysis } less morbid operation
    - can be a tough dissection (~15% conversion rate)
- other procedures
    - excision + reanastomosis, Boari flap, ileal ureter, auto-Tx, Nx

Complications of ureterolysis

- **ureteric injury (most common)**
- urine leak
- ureteral strictures
- bowel obstruction } more common if omental wrap performed
- bowel injury
- bleeding
- infection

- **long term f/u after medical or surgical therapy is essential**
  - renal fxn
  - findings suggestive of malignancy
  - recurrence or progression

- **optimal management for aortic aneurysm associated with RPF**
  - AAA repair + ureterolysis normalized renal function in 75%
  - endovascular repair (EVAR) alone can resolve RPF in many patients

- **multifocal fibrosclerosis syndrome**
  - syndrome characterized by fibrosis involving multiple organ systems
    - idiopathic RPF
    - sclerosing mediastinitis
    - sclerosing cholangitis
    - orbital pseudotumour
    - Riedel’s thyroiditis

Pelvic Lipomatosis

It is a rare benign condition marked by pelvic overgrowth of non-malignant but infiltrative adipose tissue

Incidence **راجل اسود تخيين في الاربعينات**

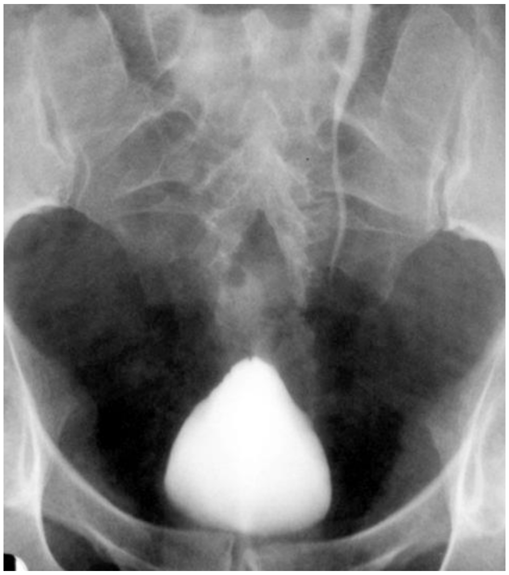
- mean age at presentation is 40yrs
- mostly in **males**
- more common in **blacks**
- associated with **obesity** but unknown etiology

Presentation **“**mostly nonspecific”

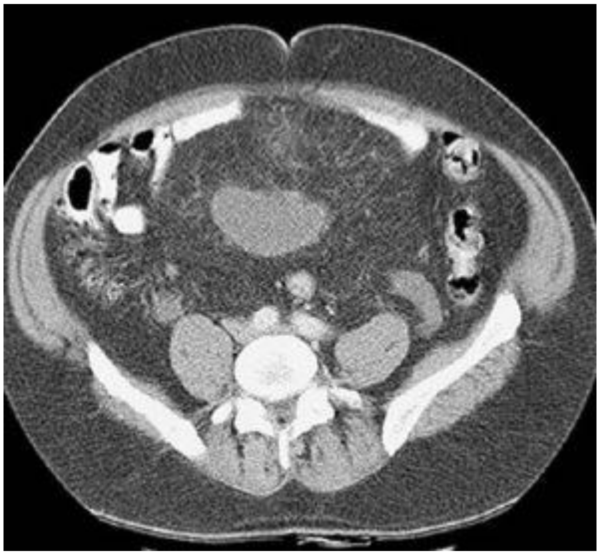
- **LUTS (50%)** or constipation (25%)
- suprapubic mass, high-riding prostate, indistinct pelvic mass
- 25-75% have **HTN**
- Rare
  - DVT
  - renal failure due to obstruction

Radiologic investigation

- KUB
  - increased pelvic lucency
- IVP
  - pear-shaped bladder with surrounding pelvic lucency
  - elevated bladder base
  - medial deviation of ureters
  - hydroureteronephrosis (found in 1/3)



- CT **(Diagnostic modality of choice)**
    - increased pelvic fat
    - extrinsic compression of bladder and rectum
    - R/O liposarcoma if there is heterogeneously enhancing regions of fat



- VCUG
    - elongated posterior prostate
    - anterior displacement & elevation of bladder

Management

1) evaluation

- cystoscopy
  - consider Bx in all patients, even if asypmtomatic as cystitis cystica and **cystitis glandularis found in 40%**
    - surveillance if found as risk of **adenocarcinoma of bladder**
  - rigid cystoscopy may be difficult due to elongation of prostatic urethra, high BN, & fixed pelvis
- consider Bx of tissue if suspicious for liposarcoma (linear areas of increased attenuation)

2) Treatment

- weight loss (controversial)
- NOT EFFECTIVE } steroids, Abx, RADs
- long term f/u is mandated } risk of progressive ureteral obstruction & risk of bladder adenocarcinoma
- surgical removal of fat or any other pelvic surgery VERY HARD
  - obliteration of surgical planes
  - increased vascularity within fatty mass
- TURP also difficult due to elongated urethra
  - may need perineal urethrostomy

DD OF causes of a pear-shaped bladder

- Urinoma , Hematoma , Abscess
- Pelvic lipomatosis
- Neoplasm (liposarcoma, lymphoma, etc)
- Radiation fibrosis
- Aneurysm (common iliac artery)
- Cement extrusion from THR
- Psoas hypertrophy

hydronephrosis during pregnancy

- more common during first pregnancy
- found in 40-100% of pregnancies
  - degree of hydro increases with time 15% in T1, 20% in T2, **~90% in T3**
- more common on R side
  - engorged uterine vein, derotated gravid uterus
  - L side protected from compression by sigmoid
- usually resolves within 6 weeks post-partum
- etiology
  - hormonal (progesterone)
  - mechanical (uterus)
- presentation
  - usually clinically silent can develop flank pain, pyelonephritis
- investigation
  - U/S
    - hydronephroureterosis down to level of pelvic brim (if lower, think stone)
    - Doppler U/S can look for ureteric jets
  - MRI
    - can see filling defects within distal ureter
    - can also r/o appendicitis, ovarian torsion, adrenal hemorrhage
- management of symptomatic patient only
  - iv fluids, analgesics, Abx (as needed) for most case
  - may need ureteral stent or NT placement rapid encrustation of stents during pregnancy from increased urine Ca excretion

Benign Pelvic Abnormalities that can cause extrinsic ureteral compression

- **tubo-ovarian abscess**
  - occurs in **15% of women with PID**
  - Rx
    - open or lap excision or transvaginal drainage
    - may need NT or stent for high-grade obstruction or urosepsis
- **endometriosis**
  - usually involves the **bladder** (80%) but ureter is involved in ~15-20%
  - usually **extrinsic** involvement but can also be intrinsic
  - usually involves **distal ureter , more common on L side**
  - **ureterosacral ligament most common site of ureteric obstruction**
  - most w/ ureteral endometriosis are asymptomatic
  - Rx
    - OCP/Lupron if N renal fxn, minimal hydro, & no obstruction on renal scan
    - ureterolysis for extrinsic compression
    - distal ureterectomy + reimplantation for intrinsic disease
- **ovarian remnants**
  - syndrome results from incomplete BSO } becomes functional and cystic which lead to extrinsic ureteral obstruction
    - Rx
      - surgical excision of mass + ureterolysis
      - leuprolide for poor surgical candidates
- **mass lesions of the uterus & ovaries**

Vascular Causes of Ureteral Obstruction

A. arterial causes of ureteral obstruction

1) AAA

- can see lateral or medial deviation of ureters
- etiology of obstruction
  - extrinsic compression from mass effect
  - localized inflammation
- **obstruction usually associated with medial deviation of ureters** especially with inflammatory AAAs (only 10% of AAAs)
- Rx – stent insertion
    - open AAA repair resolves obstruction in ~80%
    - endovascular repair resolves obstruction cf 50% with

2) iliac artery aneurysms

- extrinsic compression from mass effect or localized inflammation
- 35% w/ **common iliac artery aneurysm**; 20% with **internal iliac artery aneurysm**
- suspect iliac artery aneurysm if pulsatile mass on DRE
- Rx
  - ureteral stent insertion; removed 6-8 weeks post
  - for common iliac ureterolysis OR resection of aneurysm + graft OR endovascular graft placement
  - for internal iliac : open surgical ligation + ureterolysis OR endovascular graft

3) post-graft placement

- ureteral obstruction found in **10-20% of patients after AAA repair**
- usually present in 1st post-op yr
- etiology
  - graft placement anterior to ureter
  - ureteral entrapment in perigraft fibrosis,
  - ureteral devascularization
  - unidentified ureteral ligation
  - ureteral compression from post-op pseudoaneurysm
- Rx
  - If early hydronephrosis without renal dysfunction conservative management as most resolve spontaneously some may need temporary NT or stent
  - trial of steroids or tamoxifen if from perigraft fibrosis
  - transposition of graft if graft anterior, rather than ureter (due to risk of infection),
  - ureterolysis, ileal ureter substitution, chronic ureteral stenting

B. venous causes of ureteral obstruction

1) puerperal ovarian vein thrombophlebitis

- **(2-3 days post-partum)**
- more common on R side
- abdo/flank pain, fever, and tender, indurated adnexal mass (50%)
- Rx – conservative therapy with ABx
  - stent as needed
  - lap or open ureterolysis + ovarian vein resection if no resolution

2) testicular vein thrombophlebitis

  - reported on the L side
  - Rx – ureterolysis + excision of thrombosed vein

3) retrocaval or circumcaval ureter

URETERAL STRICTURE DISEASE

causes of ureteral strictures

- idiopathic
- acquired
    - ureteral stone (impacted)
    - endoscopic instrumentation
    - infection (eg TB, schisto)
    - ischemia/trauma from surgical dissection
    - RADs
    - malignancy · TCC, metastatic cancer (eg cervical, prostate, etc)
    - periureteral fibrosis from AAA or endometriosis (ie RPF)

presentation and investigation as usuall

management

- **indications to treat a ureteral stricture**
    - pain
    - recurrent pyelonephritis
    - compromised renal function
    - to rule out malignancy

- **line of management**
    - endoscopy
    - open

1- endourologic management

contraindicated if active infection or stricture >2cm (not including stent)

A. stent

- for acute decompression
- chronic stent for patient with poor prognosis and not candidate for repair

B. balloon dilation

- retrograde or antegrade
- ~65% success rate
    - BETTER if iatrogenic non-anastomotic strictures
    - poor results with long, ischemic strictures

C. endoureterotomy

- site
    - anterior incision for lower & mid ureteric strictures (iliacs)
    - lateral/posterolateral incision for upper ureteric strictures (great vessels)
- technique
    - retrograde, antegrade, combined (cut to light)
    - cold knife, Holmium laser, cutting electrode } full thickness 2-3mm of normal ureter

2- **open surgical management**
    a. **UU**
    b. **reimplant } +/- Psoas Hitch } +/- Boari flap**
    c. **TUU**
    d. **ileal ureter**
    e. **auto-Tx**

in each indication ,contraindication ,technique, complication

1) UU

- good for **short upper ureteric or mid-ureteric** strictures
- dissect and mobilize enough ureter to make tension-free anastomosis
  - with gunshot injury, excise wider to account for blast effect
- >90% success rate

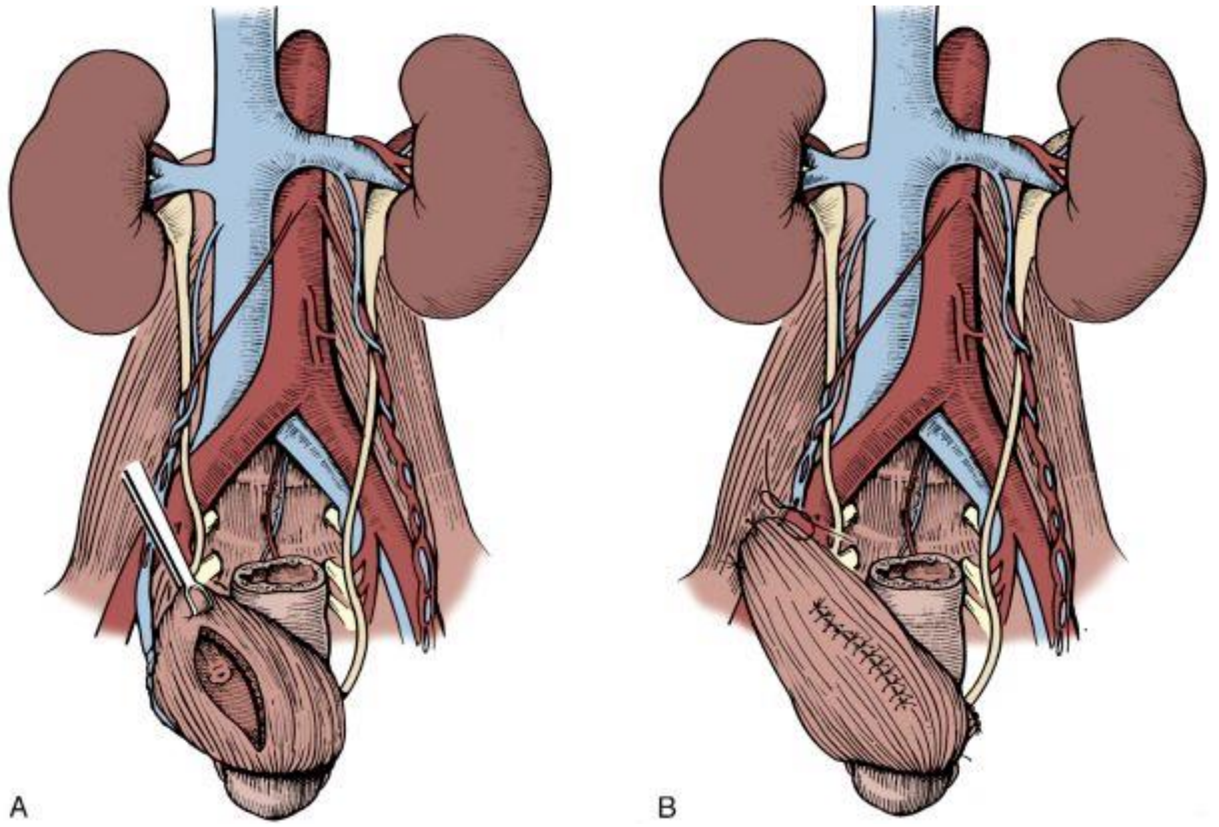
2) reimplant

- good for **distal ureteric strictures (3-4cm)**
  - can be combined with Boari flap or Psoas hitch to add length
- direct, nontunneled anastomosis or submucosal tunneled anti-reflux anastomosis
  - in adults, no difference in preservation of renal fxn or risk of stenosis
- risk of pyelo unclear

3) Psoas Hitch

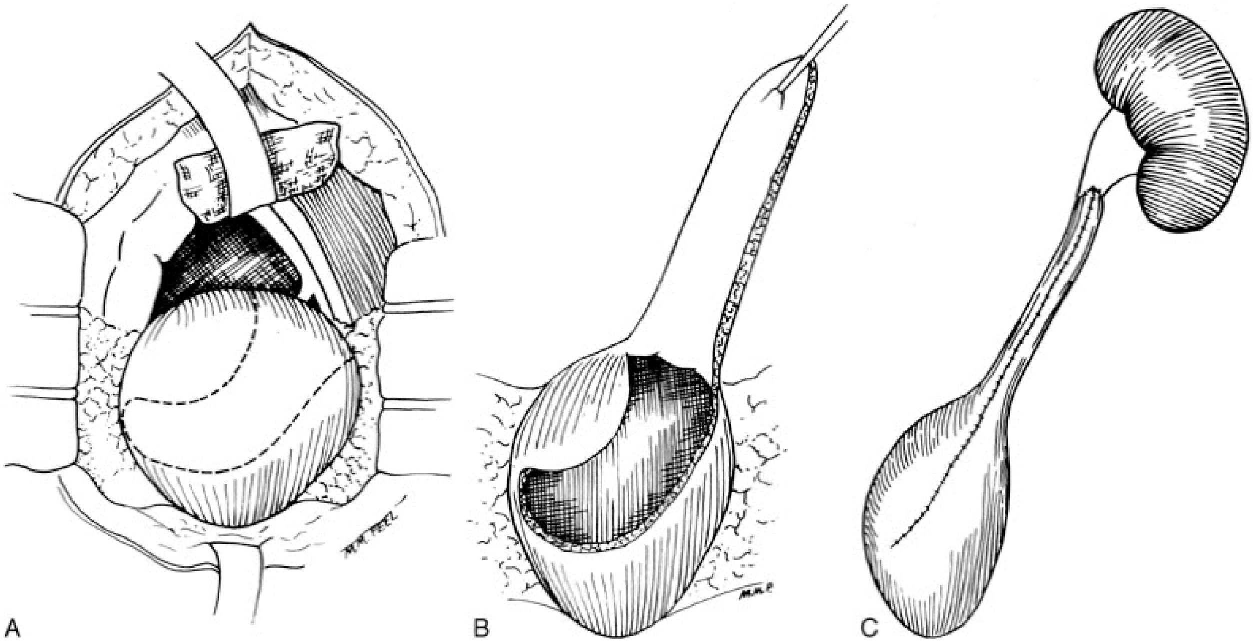
- good for **defect in distal 1/3 of ureter (up to level of pelvic brim)**
  - adds 6-10cm of length relative to simple reimplant
- **CONTRAINDICATED if small, contracted bladder w/ limited mobility**
  - need to evaluate bladder pre-op } treat BOO & neurogenic bladder

- technique
  - free peritoneal attachments and divide vas deferens/round ligament
  - for more mobility, **divide contralateral superior vesical artery**
  - ipsilateral bladder dome secured to Psoas tendon/muscle watch for **genitofemoral nerve & femoral nerve**
  - ureter inserted in anterosuperolateral position of bladder
- >85% success rate



4) Boari flap

- Good for **distal, mid & some proximal ureteric defects**
  - able to bridge 12-15cm ureteral defect
  - spiral flap can reach renal pelvis sometimes, esp on R side
- **CONTRAINDICATED if small, contracted bladder w/ limited mobility**
  - need to evaluate bladder pre-op } treat BOO & neurogenic bladder first
- technique
  - posterolateral bladder flap made based on **vascular supply of ipsilateral superior vesicle artery**
    - base should be at least 4cm, and tip at least 3cm
    - flap length should be enough to cover defect (**3:1** length:width ratio)
- recurrent strictures form due to ischemia or excessive tension



5) **TUU**

- good for **mid to lower ureteric defects + length insufficient to reach bladder**
- **contraindication**
    - **ABSOLUTE CONTRAINDICATIONS**
        - ureter too short to reach contralateral side
        - diseased recipient ureter
    - **RELATIVE CONTRAINDICATIONS**
        - stone disease,
        - RPF,
        - urothelial Ca,
        - chronic pyelo,
        - abdomino-pelvic radiation
- technique
    - mobilize colon medially on both sides,
    - expose recipient ureter ~5cm proximal to level of divided ureter, tunnel **under sigmoid colon mesentery proximal to IMA**,
    - anteromedial ureterotomy made,
    - stent placed from kidney of donor side into bladder across anastomosis
    - minimize recipient ureter dissection to maintain maximal vascular supply

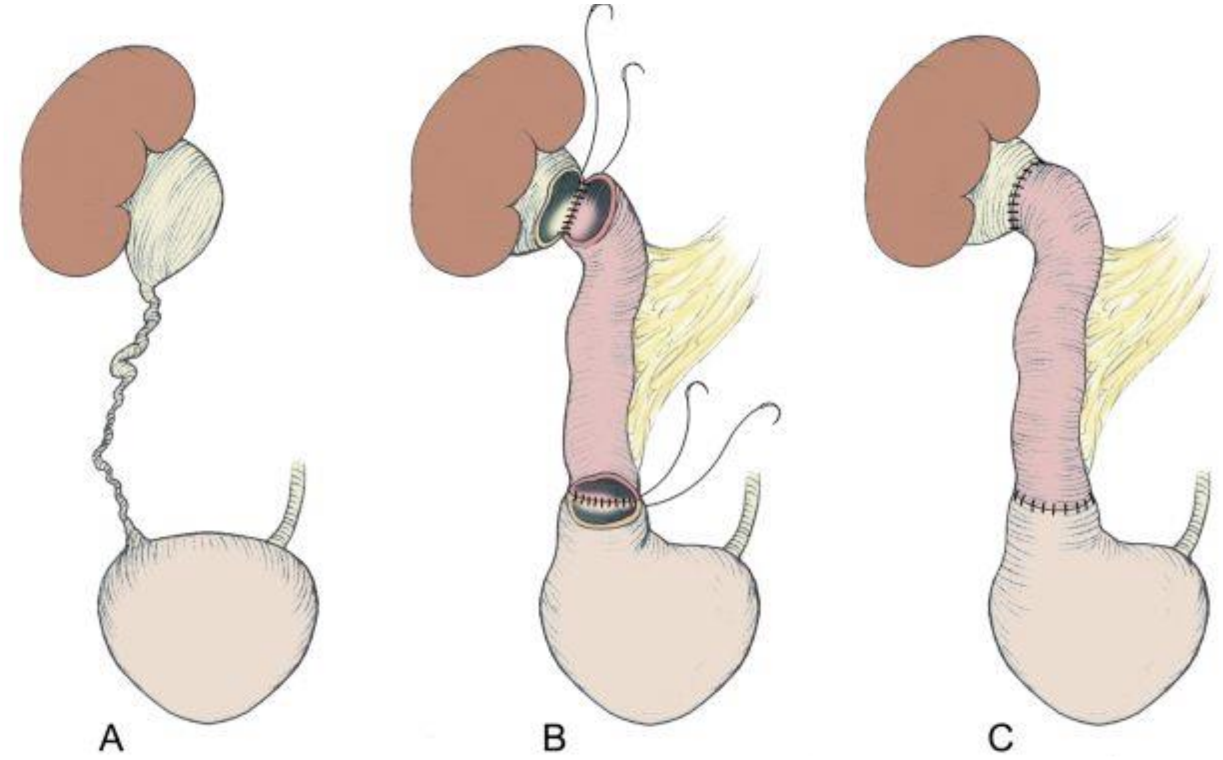
6) **intubated ureterotomy** • historical procedure no longer used

7) renal descensus

- good for **upper ureteric defects & to reduce tension on ureteral repairs**
  - able to gain 8cm of length } renal vein usually limits descensus
- technique
  - kidney mobilized completely and rotated inferiorly and medially
  - LP of kidney secured to retroperitoneal muscle

8) ileal ureteral substitution

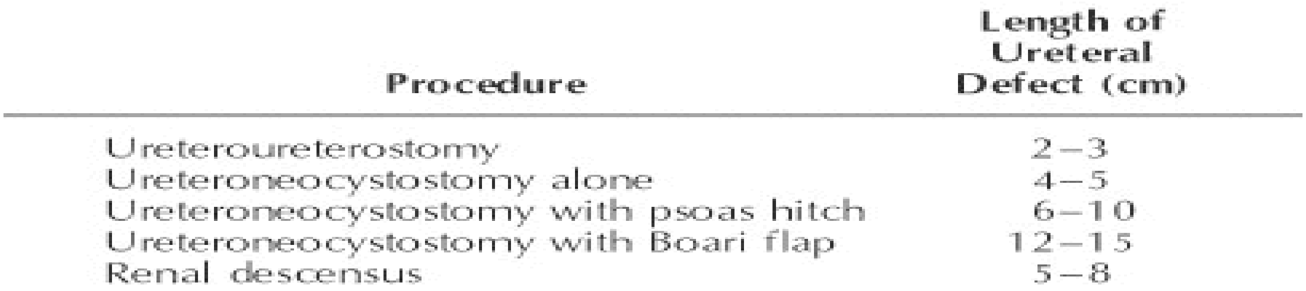
- good for **ureteral defects too long for other methods + bladder not suitable for reconstruction**
- **CONTRAINDICATIONS**
  - **renal insufficiency**
  - **bladder dysfunction or BOO**
  - **radiation enteritis**
  - **IBD**
- Technique
  - mobilize ipsilateral colon medially, measure appropriate length of ileum (15cm from ileocecal valve), divide ileal segment, deliver segment laterally through window in colonic mesentery,
  - Ensure isoperistalsis with ureter & anastomose
- electrolyte abnormalities not common if normal renal function
  - **hyperchloremic metabolic acidosis possible**
- ureteroscopy of ileal segment recommended starting 3yrs post-op to survey for **malignancy**
- **Yang-Monti** procedure also option less small bowel used



9) auto-Tx

- for **extensive ureteric defects +/- absent/poorly fxn’ing contralateral kidney**
- can perform nephrectomy laparoscopically

10) Nephrectomy



Procedure	Length of Ureteral Defect (cm)
Ureteroureterostomy	2–3
Ureteroneocystostomy alone	4–5
Ureteroneocystostomy with psoas hitch	6–10
Ureteroneocystostomy with Boari flap	12–15
Renal descensus	5–8

principles of ureteric anastomosis

- excise devitalized tissue
- limit handling of ureteric tissue
- preserve adventitia to maximize ureteral blood supply
- spatulate widely } if dilated, may transect obliquely to match caliber of lumen
- watertight, tension-free anastomosis
- stent ureter
- if possible cover with retroperitoneal fat or omentum
- place surgical drain (JP)

Ureteric stent كلام رغى

indications for ureteral stent placement.

- obstruction
    - associated with pyelo
    - associated with renal failure
    - solitary kidney
    - bilateral ureteral obstruction
    - refractory renal colic from obstruction stone
    - palliation of renal colic during pregnancy
- promotion of ureteral healing
    - minor perforation
    - major transection, avulsion (repair + stent)
    - persistent urinary extravasation after renal trauma
    - post-op } UPJO repair, ureteral stricture repair, ureteroenteric anastomosis,
- ureteric reimplantation
    - renal Tx
- prophylactic measure
    - prior to SWL ("SORRI, Stent Patient Before" • eg Solitary, Obstruction, Renal colic, etc)
    - pre-URS to allow passive dilatation (eg paeds)
    - post-complicated URS } not necessary for routine, uncomplicated URS
    - pre-op to assist with intra-op identification of ureter (eg gyne surgery)
- upper tract access
    - upper tract BCG (stent + bladder instillation)

complications

- intraop
  - ureteral injury
  - stent migration (usually due to inadequate length)
- early post-op
  - hematuria
  - stent colic
  - storage LUTs
  - stent migration
- late post-op
  - UTI
  - stent encrustation (most important predictor is indwelling time)
  - stent fragmentation
  - forgotten stent

properties of ideal stent

1. **physical of an**
   - high tensile strength
   - low coefficient of friction
   - high degree of radiopacity
   - durometer
   - high elasticity
   - biocompatible
   - biodurable

2- clinical

- easy to insert
- easy to restore & maintain antegrade flow
- resists migration
- minimal stent symptoms
- cost-effective
- radiopaque
- resist encrustation & biofilm
- biocompatible & biodurable